Dissertation on

A CLINICAL STUDY ON ANTERIOR ISCHAEMIC OPTIC NEUROPATHY

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CERTIFICATE

This is to certify that this dissertation entitled **"A CLINICAL STUDY ON ANTERIOR ISCHAEMIC OPTIC NEUROPATHY"** is a bonafide record of the research work done by **Dr. VINAYA,** post graduate in Regional Institute of Ophthalmology and Government Ophthalmic Hospital, Madras Medical College and Government General Hospital, Chennai-03, in partial fulfillment of the regulations laid down by The Tamil Nadu Dr.M.G.R. Medical University for the award of M.S. Ophthalmology Branch III, under my guidance and supervision during the academic years 2011-2014.

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Clinical study on anterior ischaemic optic neuropathy

Abstract

A prospective observational study was conducted over a period of 12 months on 35 patients presenting with typical features of anterior ischaemic optic neuropathy. Patients with infectious/inflammatory diseases/other disorders that could cause visual defect were excluded from the study. It was found that this disease mostly affects the elderly population with 51-60 years being the most affected age group. Females were found to be more affected than males with the predominance of right eye over left eye. Diabetes mellitus was found to be the most common associated risk factor followed by systemic hypertension. 69.44% of the subjects presented with poor visual acuity in the range of 2/60 to 6/60. Pallid disc oedema was the most common ophthalmoscopic finding followed by sectoral disc pallor. Superior and inferior altitudinal field defects were predominant among patients in whom automated perimetry was possible. In most of the patients, vision remained less than 6/60 despite timely intervention with corticosteroids. The presence of diabetes mellitus is associated with poor visual outcome. But hypertension and hyperlipidemia did not affect the visual outcome. Thus anterior ischaemic optic neuropathy must be considered as an important differential diagnosis of painless loss of vision in elderly population associated with optic disc oedema. All patients must be thoroughly evaluated for systemic risk factors and promptly treated to prevent the condition in the other eye

Keywords – anterior ischaemic optic neuropathy, pallid disc oedema, sectoral pallor, altitudinal visual field defect, corticosteroids, automated perimetry.

CONTENTS

ABBREVIATIONS

RE : RIGHT EYE

LE : LEFT EYE

NAD : NO ABNORMALITY DETECTED

DM : DIABETES MELLITUS

HTN : HYPERTENSION

AAION : ARTERITIC AION

NAION: NON ARTEITIC AION

ESR : ERYTHROCYTE SEDIMENTATION RATE

FDT : C REACTIVE PROTEIN

GCA : GIANT CELL ARTERITIS

ONH : OPTIC NERVE HEAD

IOP : INTRAOCULAR PRESSURE

BP : BLOOD PRESSURE

FBS : FASTING BLOOD SUGAR

PART I

INTRODUCTION

AND

HISTORY

INTRODUCTION AND HISTORY

Anterior ischaemic optic neuropathy is a common disease which affects visual acuity in the adult and elderly population. The annual incidence of NAION is approximated to be around 2.3 to 10.2 per 100,000 for people aged 50 years and above[1]. It is known to be associated with many circumstances that may influence and decrease the blood supply to optic nerve head. Some of these risk factors are diabetes, hypertension, hyperlipidemia, hyperhomocysteinemia and pro-thrombotic disorders. It is believed to be due to circulatory insufficiency associated with blood supply of the anterior portion of optic nerve.

ANATOMY AND BLOOD SUPPLY OF THE OPTIC NERVE HEAD

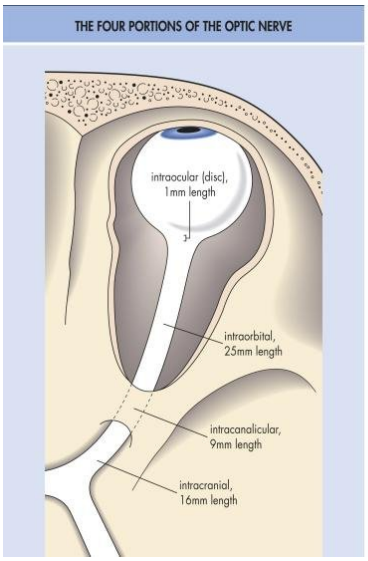


Fig 1: Picture depicting the anatomy of optic nerve

ANATOMY OF THE OPTIC NERVE

The optic nerve is the second cranial nerve which begins from the optic disc and extends up to the optic chiasma where the two nerves meet. It is the backward continuation of the retinal nerve fibre layer which consists of axons taking origin from the ganglion cells.

PARTS OF THE OPTIC NERVE

The optic nerve which is about 47-50 mm in length is divided into four parts

1. Intraocular (1mm)
2. Intraorbital (30 mm)
3. Intracanalicular (6-9mm)
4. Intracranial (10mm)

INTRAOCULAR PART

Axons of the retinal ganglion cells form bundles that constitute the nerve fibre layer which converges at the optic nerve head. It has an average horizontal diameter of 1.5 mm and vertical diameter of 1.8mm. It is a major zone of transition because nerve fibres pass from an zone of high pressure to a zone of low pressure that corelates with the intracranial pressure. The nerve fibres leave an area of blood supply from the central retinal artery to zones supplied by other branches of ophthalmic artery.

The axons also become myelinated immediately at the posterior end of optic nerve head where it expands to 3mm.[2]

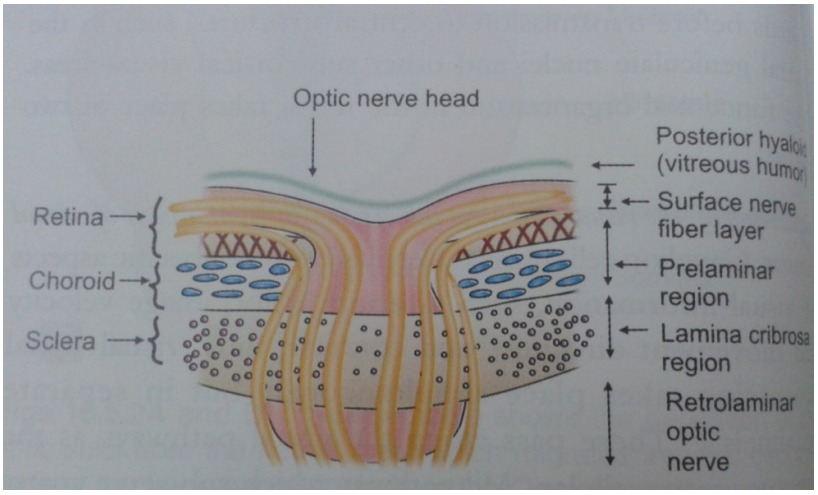


Figure 2 – picture depicting anatomy of optic nerve head

Arbitrarily the optic nerve head is divided into four portions from anterior to posterior -

1. **Surface nerve fibre layer**

   Is composed of nerve fibres of retina which converge on optic disc and astrocytes. The optic disc is covered by a thin layer of astrocyte- the internal limiting membrane of Elschnig which separates it from the vitreous and is continuous with internal limiting membrane of retina.

2. **Prelaminar region**

   The predominant structures at this level are neurons and a significantly increased quantity of astroglial tissue. The border tissue seaperates the nerve from the choroid.

3. **Lamina cribrosa**

   It is a fibrillar sieve like structure made up of fenestrated sheets of scleral connective tissue lined by glial tissue. It bridges the posterior scleral formina or the scleral canal. The bundles of optic nerve fibres leave the eye through the fenestrations. A rim of collagenous

connective tissue with some admixture of glial cells which intervenes between the choroid and sclera and optic nerve fiibres is the border tissue of Elschnig.

4. Retrolaminar region

There is a decrease in astrocytes and acquisition of myelin that is supplied by oligodendrocytes. The addition of myelin sheath nearly doubles the diameter of the optic nerve as it passes through the sclera. The axonal bundles are surrounded by connective tissue septa.

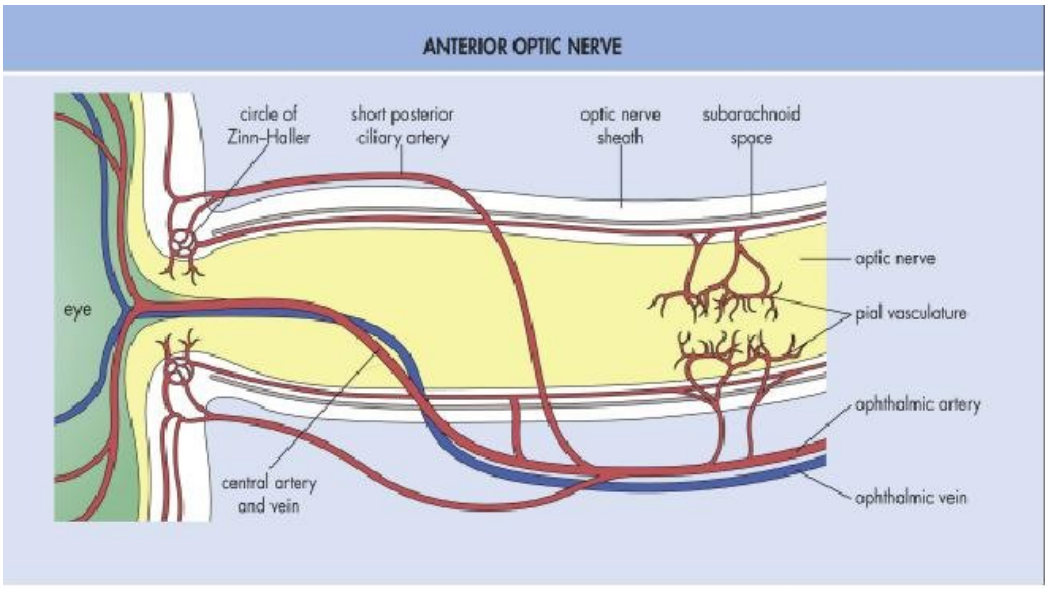


Figure 3 – picture showing the anterior part of the optic nerve

INTRAORBITAL PART

Extends from back of eyeball to the optic nerve foramina. This part is slightly sinuous to allow or eye movements.

INTRACANALICULAR PART

Closely related to the ophthalmic artery which crosses the nerve inferiorly from medial to lateral side in the dural sheath and leaves the sheath at the orbital end of the canal.

INTRACRANIAL PART

Lies above the cavernous sinus and converges with its fellow to form the optic chiasma. This part is ensheated in piamater, but recieves arachnoid and dural sheaths at the point of its entry into the optic canal.

BLOOD SUPPLY OF THE OPTIC NERVE HEAD

Optic nerve recieves its blood supply from the posterior ciliary vessels with a small contribution from central retinal vessels[3]. The source and pattern of the blood supply of anterior part (optic nerve head) differs from that of the posterior part. The optic nerve head is almost entirely supplied by the posterior ciliary artery circulation while the rest of the optic nerve posterior to it is supplied from several other sources.

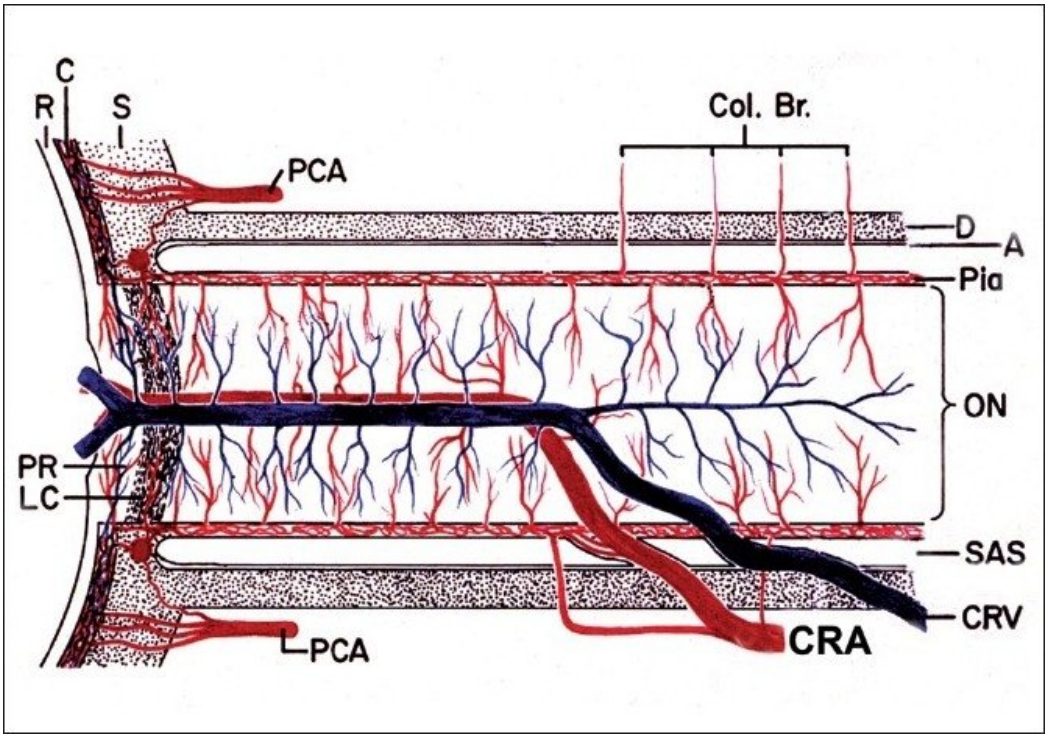


Figure 4 – schematic representation of blood supply of optic nerve

1. SURFACE NERVE FIBRE LAYER – supplied by the capillaries derived from the retinal arterioles, which anastomose with vessels of the prelaminar region. Occasionally a ciliary derived vessel from the prelaminar region may enlarge to form the cilioretinal artery.

2. PRELAMINAR REGION – suppiled by vessels of ciliary region. These vessels are derived from separate branches of short posterior ciliary arteries.

3. LAMINA CRIBROSA REGION – is also supplied by ciliary vessels. They commonly arise from short posterior ciliary arteries and also commonly the arterial circle of Zinn –Haller.

4. RETROLAMINAR REGION – it is the part that lies immediately behind the lamia cribrosa. It is supplied by both ciliary and retinal circulation with the former coming from the recurrent pial vessels. Central retinal artery provides centripetal brances from the pial plexus and also centrifugal branches.

   Axial centrifugal vascular system – it is formed by the inconstant branches arising from the intra neural part of central retinal artery. However, it is not consistently present in all nerves.

Therefore the main source of blood supply to the optic nerve head is the PCA circulation via the peripapillary choroid and the short PCAs (or circle of Zinn and Haller). Flourescein angiography studies have shown sectoral blood supply in the ONH which goes along with the overall segmental distribution of the PCA circulation which helps to explain the segmental visual loss in AION.

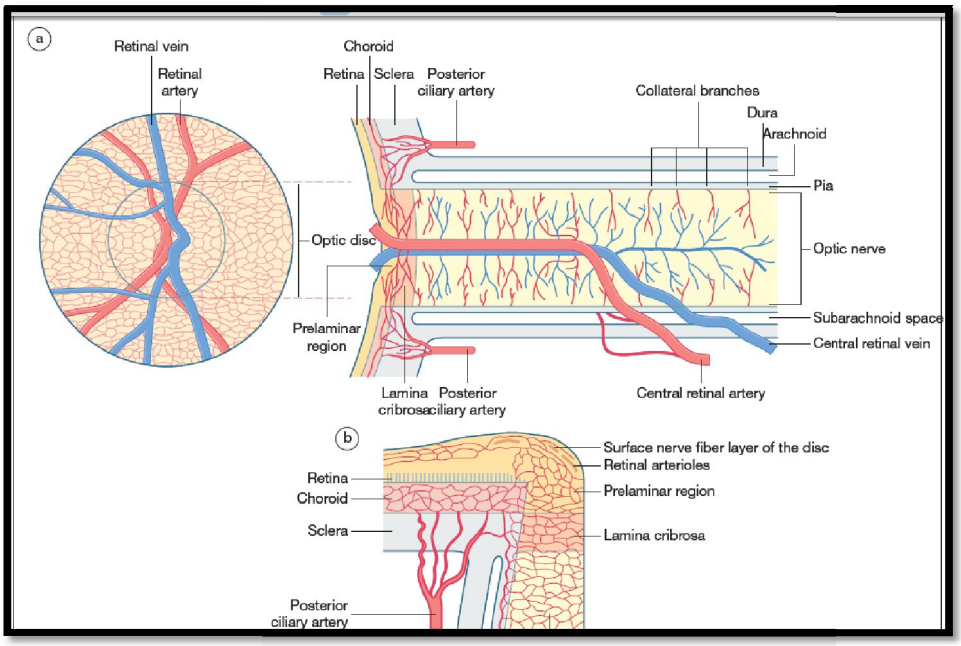


Figure 5: Picture representing the blood supply of the optic nerve head

Arterial blood supply of the posterior part of the optic nerve

This part of the optic nerve has a peripheral and an axial vascular system.

Peripheral centripetal vascular system- formed by the pial vessels which come from collateral arteries arising directly from the ophthalmic artery and some its intraorbital branches.[4]

Axial centrifugal vascular system – formed by branches of central retinal artery seen in 75% of cases and the supply of central retinal artery may extend 1 to 4 mm behind the site of penetration of central retinal artery into the optic nerve and give rise to centrifugal branches.

INTER-INDIVIDUAL VARIATION IN THE BLOOD SUPPLY OF THE OPTIC NERVE HEAD

The pattern of blood supply of the optic nerve head is not identical in all eyes. There is a marked variation among individuals which is produced by the following factors[4] :

1. Variation in anatomical pattern of the arterial blood supply

2. Variations in the Posterior ciliary artery circulation – there may be between one to five PCAs but usually 2 or 3 PCAs are usually present. The PCAs enter the eyeball usually medial and

lateral to the optic nerve and hence they are called as medial and lateral PCAs.

Hayreh et al[5] have shown that PCAs and their branches have segmental distribution in vivo in the choroid as well as in the ONH. The medial PCA may supply the entire ONH or it may take no part in the blood supply of the ONH and there may be any number of variations between these two extremes. The lateral PCA supplies the area of ONH not supplied by the medial PCA or vice versa. When there is more than one medial or lateral PCA, the area is supplied by each and may be only a sector. When the superior PCA is present, it accordingly supplies a superior sector. Therefore the inter individual variation in number and distribution by the various PCAs produces an extremely variable pattern of distribution by the PCAs in the optic nerve head. This is very important while dealing with ischemic disorders of the ONH.

WATERSHED ZONES IN THE PCA DISTRIBUTION AND THEIR LOCATION –

When a tissue is supplied by two or more end-arteries, the border between the territories of distribution of any two end arteries is

called a ‘watershed zone’. The significance is that in the event of a fall in the perfusion pressure in the vascular bed of one or more of the end arteries, the watershed zone being an area of comparatively poor vascularization is most vulnerable to ischaemia. Since PCAs and their subdivisions are end arteries in vivo they have watershed zones between them.

Ischaemia of the anterior part of optic nerve most commonly occurs where there is crowding of nerve fibres and decrease in blood supply may merge to decrease perfusion to a severe extent.

The blood flow within the optic nerve head depends upon several factors, the most important of which is blood pressure within the vessels.

The blood flow in the ONH is calculated by using the formula[6] –

Perfusion pressure = Mean BP – intraocular pressure

Mean BP = diastolic BP + 1/3 (systolic – diastolic BP)

Therefore, blood flow depends upon – resistance to blood flow, BP and intraocular pressure.

Thus, a reduction in blood flow may develop consequent to any of the three factors. Transient poor circulation or loss of circulation in the optic nerve head can occur due to a transient fall of blood pressure below a critical level in its vessels, which in turn in susceptible persons could produce AION[7].

Normally there is an auto regulation mechanism that operates in the optic nerve and helps to compensate for any decrease for any decrease in the blood flow, but it operates only over a critical range of perfusion pressure so that with a rise or fall of perfusion pressure beyond which this mechanism fails.

Factors leading to the derangement of auto regulation in the ONH include systemic and local factors[8], aging, arterial hypertension, diabetes mellitus, marked hypotension due to any cause, atherosclerosis, hypercholesterolemia, arteriosclerosis and hyperhomocysteinemia.

ANTERIOR ISCHAEMIC OPTIC NEUROPATHY

It is an optic neuropathy of acute or sub-acute onset which occurs due to precarious decrease in blood flow to axons of retinal ganglion cells. It is a widespread visually disabling disorder that is commonly seen in the middle aged and elderly population. The classical presentation involves sudden loss of a part of the visual field.

There are two clinical types –

1. Arteritic AION
2. Non arteritic AION

ARTERITIC AION

This type is less common than the non-arteritic type. It is most commonly associated with Giant cell arteritis due to occlusion of SPCA leading to decrease in blood flow to proximal part of the optic nerve and choroid. Inflammatory response occurs involving the medium and large sized arteries. Focal and sectoral granulomatous inflammation occurs between the tunica intima and tunica media.

Intravascular inflammation leads to secondary obstruction of the blood vessel lumen due to occlusion.

Giant cell arteritis commonly occurs in the elderly population more than 50 years of age with a female predilection. It is a systemic disease process which presents with – jaw claudication, headache, scalp tenderness, weight loss, fatigue, night sweats and polymyalgia rheumatica[10].

Vision loss is usually severe with visual acuity <6/60 at presentation. Some patients experience transient loss of vision preceding the attack. On examination, patients usually have pallid optic disc oedema. There may be cotton wool spots suggestive of co-existing ischaemia in the retina. The optic disc of other eye in arteritic type is most commonly of normal size with a normal cup. In the non arteritic type, the optic disc is smaller in size with a small or no cup[11].

In suspected cases of AAION, erythrocyte sedimentation rate along with platelet count must be ordered. A rise in ESR and CRP has the greatest specificity for diagnosis of temporal arteritis.
A definitive diagnosis can be made with temporal artery biopsy.

Risk of involvement of other eye varies from 54 to 95%. Time to second eye involvement varies from hours to days. Optic disc oedema

resolves over four to eight weeks with disc pallor and generalized attenuation of arterioles in the posterior pole.

NON ARTERITIC AION

This entity is more common than AAION. There is higher incidence in white population and no gender predilection. Though the exact etiology remains unknown, it is thought to be due to stenosis of blood vessels supplying the proximal part of the optic nerve – direct branches of PCA and circle of Zinn-Haller.

Small optic disc with a small cup-to-disc ratio creates a crowding which causes a compromise in vascular microcirculation. NAAION is known occur in patients[12] with sleep apnoea, nocturnal hypertension, vasculopathic systemic diseases and prothrombotic factors. Other known risk factors are systemic hypertension, diabetes mellitus, hypercholesterolemia, anaemia.

Ocular risk factors commonly associated include – hypermetropia, optic disc drusen, elevated intraocular pressure, presence of disc oedema.

Vision loss is less severe than with AAION. Patients present with unilateral painless visual loss developing over few hours or days. Colour vision loss tends to parallel vision loss. Any type of visual field defects may occur, but inferior altitudinal field loss usually occurs in majority of the patients.

On examination, optic disc oedema maybe diffuse or sectoral with prominent vasculature and haemorrhages. Pallor of optic disc is less common than in arteritic form. The optic disc in the fellow eye is observed to be lesser in diameter and exhibits a small or deficient cup –"disc at risk".

Recurrence of NAION in the affected eye occurs in less than 5% of cases. In the IONDT approximately 15% of patients developed the condition in contra lateral eye within a period of five years[13].

HISTORY AND EVALUATION

HISTORY AND EVALUATION

1. **Symptoms:** Defective vision, headache, presence of scalp tenderness, field defects, jaw claudication

2. **History**

   - Diabetes Mellitus, Hypertension, hyperlipidemia, anaemia, connective tissue disorder

   - H/O similar episode in the past in same or other eye

3. **Clinical Features**

AAION - visual loss is usually profound, resulting in either complete blindness or extremely poor vision. The loss of vision can occur at any time of the day, though it is most commonly observed to occur during the night.

Pupil shows relative afferent papillary defect, which can be graded using neutral density filters. Graded denomination of the neutral density filter is placed in front of the better eye and the measurement of the filter in log units which results in equalization of the pupillary reflexes gives the numerical magnitude of RAPD.

Colour vision maybe difficult to assess due to poor vision. But in eyes with better visual acuity, it may be observed to be defective.

On ophthalmoscopic examination, during the initial stages there is optic disc oedema which is more in one part of the disc than the other. Frequently there are splinter haemorrhages at the disc margin. According to a study by Hayreh et al, in 69% of arteritic AION eyes, the optic disc has chalky white appearance[14]. Gradually the disc swelling progresses to pallor within two to three months.

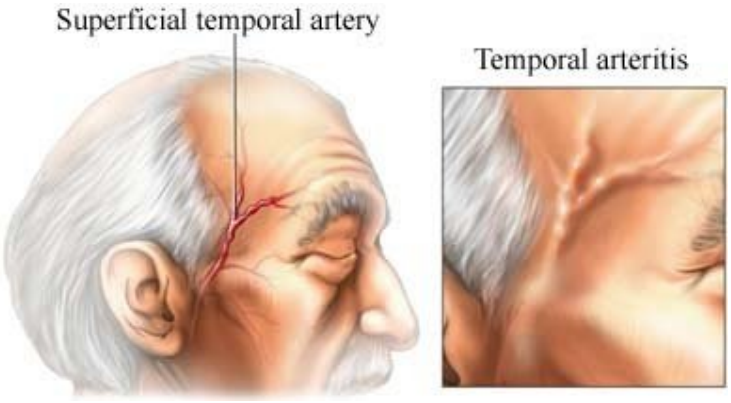


Figure 6 – picture showing surface marking of superficial temporal artery

NA-AION – it usually begins with sudden and painless deterioration of vision, which may progress rapidly over few hours to days. It is usually reported by the patients on waking up in the morning and classical presentation is involvement of lower visual field.

Pupil shows relative afferent papillary defect. Ophthalmoscopy shows an oedematous disc initially with a classic appearance of pallid disc oedema. There is no correlation between the extent and severity of optic disc

pallor and severity of visual loss. The fellow eye typically shows no cup or a very small cup.

Figure 7 – Fundus photographs showing optic disc oedema progressing to disc pallor in a patient with NAAION

OTHER INVESTIGATIONS

FUNDUS FLOURESCEIN ANGIOGRAPHY – in non arteritic AION, during the very early stages of the disease, angiography may show filling defects in the optic disc. In contrast, peripapillary choroidal filling is not delayed.

In arteritic AION, this test is extremely helpful in making the diagnosis because it shows both the choroid and the optic disc in the area supplied by the involved posterior ciliary artery do not fill.

AUTOMATED PERIMETRY – By Octopus to look for altitudinal field defects, the progression of which can be monitored during the follow up.

BLOOD INVESTIGATIONS –

Complete haemogram including Erythrocyte sedimentation rate is routinely recommended while investigating patients with ischemic optic neuropathy. ESR tends to increase with age. Miller and Green have provided a rule for calculation of normal maximum ESR at a given age –

Men – Age in years /2

Women – Age in years +10 / 2

According to a study by Hayreh et al, cut off criteria of 33 mm/1st hour in men and 35 mm/1st hour in women may provide a sensitivity and specificity of 92%.

The same study suggested that the sensitivity and specificity of C-reactive protein in detecting cranial arteritis were 100% and 83 % in men and 100% and 79% in women respectively.

Random blood sugar and lipid profile are also recommended to assess etiological associations and for monitoring the treatment.

ROLE OF TEMPORAL ARTERY BIOPSY

This is useful to make a definitive diagnosis of giant cell arteritis. It is recommended that biopsy should be done in every case of suspected AAION as treatment necessitates high dose of corticosteroids for a longer time. It is known that around five percent of temporal artery biopsy specimens may be false negative. Reason for this may be partial treatment, inadequate sample, or presence of skip lesions. Therefore a negative biopsy does not exclude presence of temporal arteritis. So, in cases of strong suspicion, sequential, and bilateral biopsies may be performed. Generally 2 to 3 cm biopsy specimen is obtained to avoid missing diagnosis due to skip lesions.

It is recommended that treatment with high dose corticosteroids should be started immediately before biopsy is performed. However, the biopsy must be done within one to two weeks following the beginning of therapy because treatment may decrease the inflammation in the biopsy specimen rapidly. Histopathological staining of the internal elastic lamina may yield additional sensitivity by demonstrating the features of non atherosclerotic wall disruption.

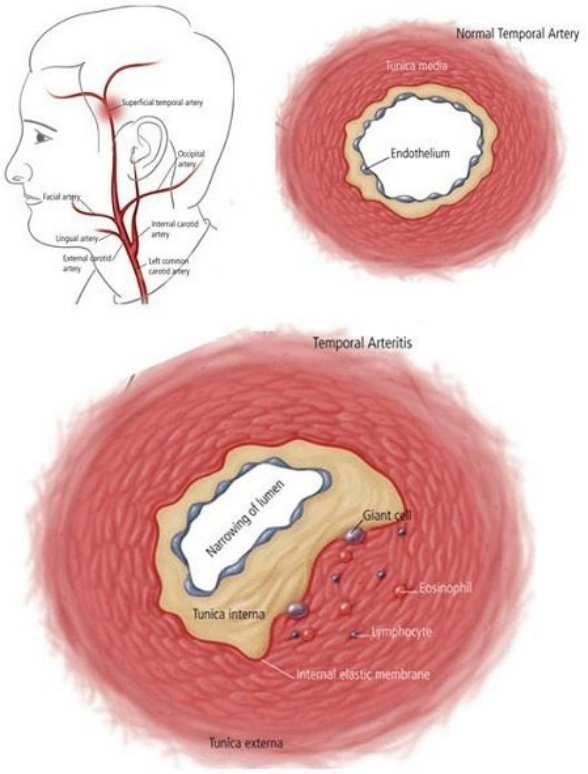


Figure 8 - schematic picture showing the pathology of giant cell arteritis.

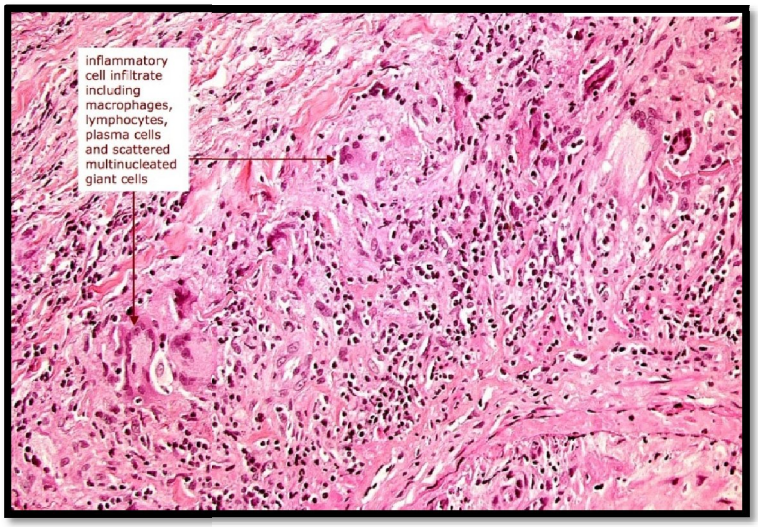


Figure 9 - Histopathology of temporal artery biopsy in giant cell arteritis.

MANAGEMENT

Visual prognosis for patients with ischaemic optic neuropathy does not change much with any form of treatment. Prior to randomized controlled trials, optic nerve decompression was believed to be useful in patients. The role of systemic steroids has also not been clearly established. Most physicians like to give a trial of systemic steroids in patients with acute presentation of NA AION. Prognosis for visual recovery is usually poor, although occasionally some patients may show good visual recovery also[15].

On the contrary, the mainstay of treatment in patients with giant cell arteritis is in the form of high dose of systemic corticosteroids.

Goal of management in patients with ischaemic optic neuropathy is –

-to establish the diagnosis

-to distinguish between arteritic and non arteritic forms of the disease.

SYSTEMIC CORTICOSTEROID THERAPY

The most important step in all patients above 55 years of age is to rule out giant cell arteritis immediately, because it is an emergency which can be prevented with aggressive treatment.

The main objective of treatment is to prevent the loss of vision in the fellow eye. The treatment of choice for giant cell arteritis is systemic corticosteroids.

The recommended dose is 1 to 2 grams/ per day of IV Methyl prednisolone for three to five days followed by oral tablet prednisolone 80 mg daily. All patients are maintained on a high dose of oral prednisolone till both the ESR and CRP have stabilized at low levels. (usually takes 2 to 3 weeks). The CRP comes down much faster than ESR. There is gradual tapering of prednisolone. It is recommended that high doses of oral steroids should be maintained for 4 to 8 weeks and then tapered gradually as long as the patient remains symptom free and ESR is below 40mm/1st hour.

It is important to remember the side effects of long term use of steroids such as gastric ulcers, myopathies, aseptic necrosis of femur, osteoporosis and worsening of blood sugar control.

Unlike arteritic AION, there is no established treatment for non arteritic AION. Definite evidence of visual improvement has been noted in some patients with oral steroids if treated early.

Role of Aspirin - A study conducted on 131 patients asserted that aspirin prevented the occurrence of non arteritic AION in the contra lateral eye. Botelho *et al*, also established that intake of aspirin does not enhance final the visual acuity in these subjects. Likewise, a similar study did not find any association linking customary aspirin intake and occurrence of new non arteritic AION in the other eye. Non arteritic AION is not a

thromboembolic ailment, but a hypotensive disorder in most of the patients. Aspirin does not have any effect on blood pressure or arterial hypotension occurring at bed time.

The Ischemic Optic Neuropathy Decompression Trial (IONDT)[16] was a randomized, single-blind, multicenter trial sponsored by the National Eye Institute to assess the safety and efficacy of optic nerve decompression surgery in patients with NAION. Analysis of data from patients enrolled in the IONDT at 24 months of follow-up confirmed that there was no benefit from optic nerve decompression surgery.

PART - II

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

Primary Objectives:

To assess the clinical presentations and visual outcomes of Anterior Ischaemic Optic Neuropathy

1. To study the natural history of AION
2. To identify potential risk factors for AION
3. To diagnose and start treatment after systematic evaluation

Secondary objective

To assess visual outcome after treatment of Anterior ischaemic optic neuropathy

MATERIALS AND METHODS

MATERIALS AND METHODS

Subject Selection :

All cases attending Ophthalmology OPD with typical signs and symptoms of Anterior ischaemic optic neuropathy in the period of 12 months.

Inclusion Criteria:

Patients with characteristic clinical features of AION such as – sudden loss of vision, presence of relative afferent pupillary defect, optic disc oedema, and optic disc margin blurring, defective visual fields

Exclusion Criteria :

1. Patients with multiple sclerosis with or without optic neuritis

2. Presence of infectious /inflammatory disease

3. Clinical features of retinal/vitreous/ other optic nerve pathology that could cause defective vision or field changes.

REGISTRATION

NAME: AGE: SEX: M/F

OCCUPATION: ADDRESS:

EYE INVOLVED: RE/ LE

HISTORY OF THE PRESENTING ILLNESS

The common complaints were,

1. Defective vision – nature of onset, whether painless/painful, unilateral/bilateral
2. Headache
3. Associated jaw claudication/ scalp tenderness
4. Fever/ night sweats/ fatigue/ weight loss
5. Similar complaints in the past in same or other eye

Details of the progress from onset, the treatment undergone to the present state were noted.

PAST HISTORY

H/o systemic diseases like diabetes mellitus, hypertension, stroke, transient ischaemic attacks, carotid artery disease, vasculitis, ischaemic heart disease, connective tissue disorders, anaemia.

H/O any long term drug intake like nasal decongestants/amiodarone/interferon alpha was noted

PERSONAL HISTORY

Smoking, alcoholism, type of diet.

GENERAL EXAMINATION

General vital data like pulse, blood pressure, peripheral pulses were noted

OCULAR EXAMINATION

1. Induration of temporal artery region, decreased or absent temporal artery pulse, any cordlike or firmness/nodularity of temporal artery
2. Visual acuity using Snellen's chart was recorded
3. Extra ocular movements were noted both ductions and versions
4. Pupil size, shape and reaction noted
5. Anterior segment examined in detail with slit lamp.

6. A dilated fundus and refraction was done.

7. Colour vision using Ishihara pseudoisochromatic plates, intra ocular pressure measurement using Goldman applanation tonometry were done for all patients.

8. Visual fields using automated perimetry by Octopus.

INVESTIGATIONS:

1. Blood pressure
2. Complete haemogram with ESR
3. Lipid profile
4. C- reactive protein
5. Colour fundus photography
6. Fundus flourescein angiography if necessary

Complete systemic evaluation by cardiologist and physician were done in all cases to rule out systemic associations

FOLLOW UP

Recording the patients complaints whether stable/ improving / worsening

1. Visual acuity
2. Colour vision
3. Visual fields
4. Contrast sensitivity

RESULTS

RESULTS

36 eyes of 35 patients with AION were included in the study. A prospective, observational study was conducted over a period of one year.

1. AGE DISTRIBUTION

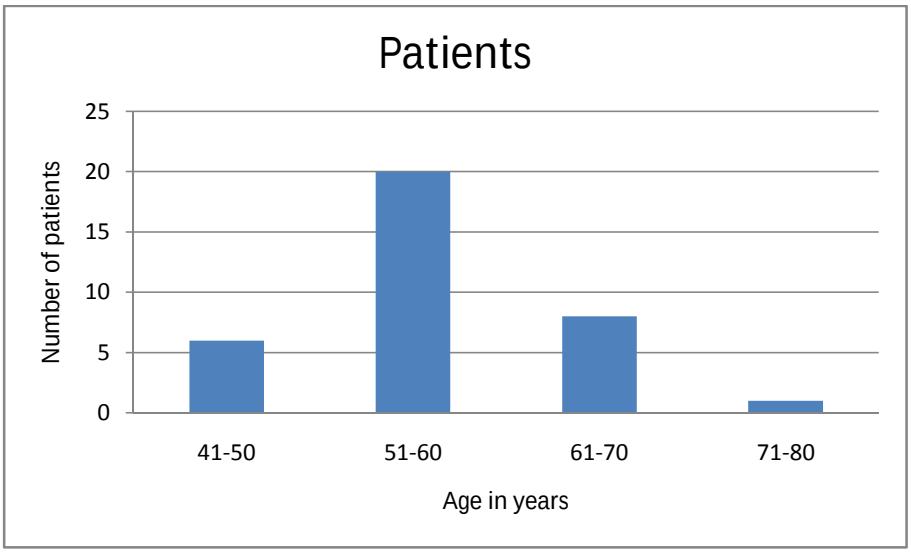
The following table shows the age distribution in patients with anterior ischaemic optic neuropathy.

TABLE – 1

Age group	**Patients**	**%**
41-50	6	17.14
51-60	20	57.14
61-70	8	22.86
71-80	1	2.86
TOTAL	**35**	**100 %**

GRAPH – 1

AGE DISTRIBUTION



In our study of 35 patients with AION, the maximum number of patients (20) belonged to the age group of 51-60 years. This constituted a percentage of 57.14%. 8 patients were from the age group of 61-70 years (22.86%) and 6 patients from age group of 41-50 years (17.14%). One patient belonged to the age group of 71-80 years (2.86%).

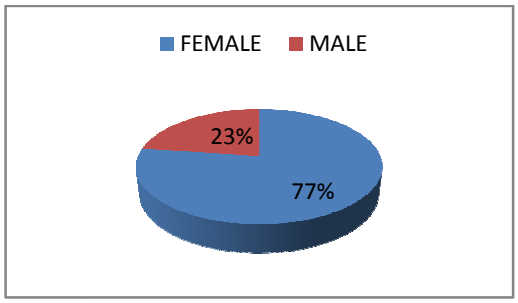
2. SEX DISTRIBUTION

TABLE – 2

	FREQUENCY	%
FEMALE	27	77.14
MALE	8	22.85
TOTAL	**35**	**100%**

In our study, there was a significant gender difference, with 27 females (77.14%) out-numbering the males 8 (22.85%).

GRAPH-2: SEX DISTRIBUTION



3. LATERALITY

TABLE-3

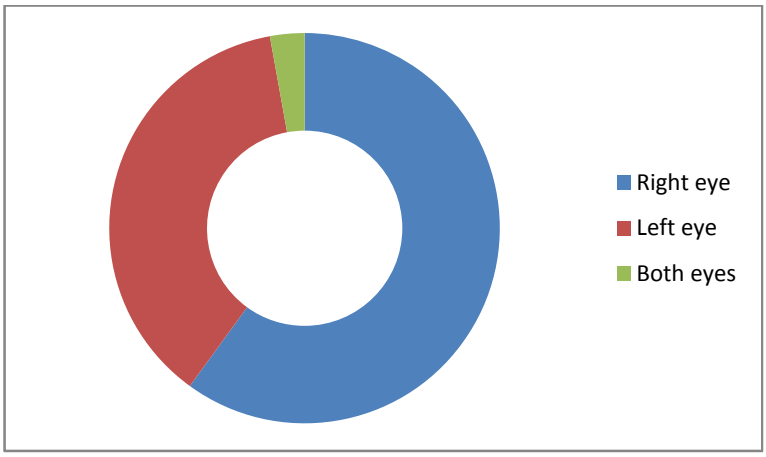
Right eye	Left eye	Bilateral	Total
21	13	1	**35**

FREQUENCY

	Frequency	%
Right eye	22	61.11
Left eye	14	38.89
Total	**36**	**100%**

In our study, there was a predominance of involvement of right eye 21 patients who constituted 60%, over left eye 13 patients who constituted 37.14%. One patient - 2.86% had involvement of other eye during the study period.

GRAPH-3: LATERALITY



4. ASSOCIATED RISK FACTORS

TABLE 5

RISK FACTOR	Frequency	%
Diabetes Mellitus	12	34.28
Systemic hypertension	9	25.71
Diabetes and hypertension	4	11.43
Anaemia	2	5.71
Glaucoma	0	0
Smoking	4	11.43
Hyperlipidemia	1	2.86
No risk factor	8	22.86

In our study, 12 patients (34.28%) who presented with AION had a history of diabetes mellitus alone. 9 patients, constituting 25.71% of the study subjects, were diagnosed patients of systemic hypertension. Out of the total 35 subjects, 4 patients (11.43%) were found to have both – diabetes mellitus and systemic hypertension. 4 subjects (11.43%) gave a history of smoking cigarettes/ beedis. Anaemia was found to be associated with 2 (5.71%) of the subjects. One patient (2.86%) had hyperlipidemia. 8 patients (22.86%) who were diagnosed with AION did not have any identifiable risk factors attributable to the condition.

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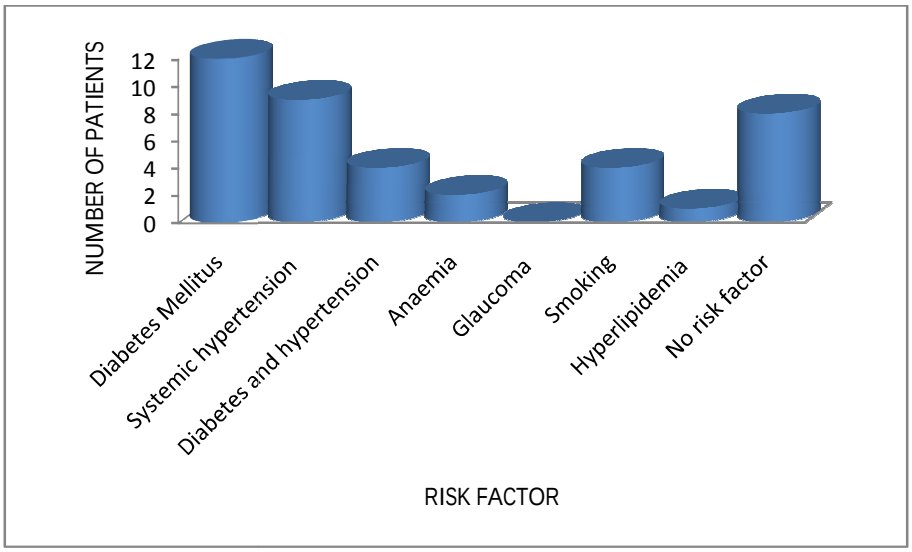
GRAPH-4: ASSOCIATED RISK FACTORS



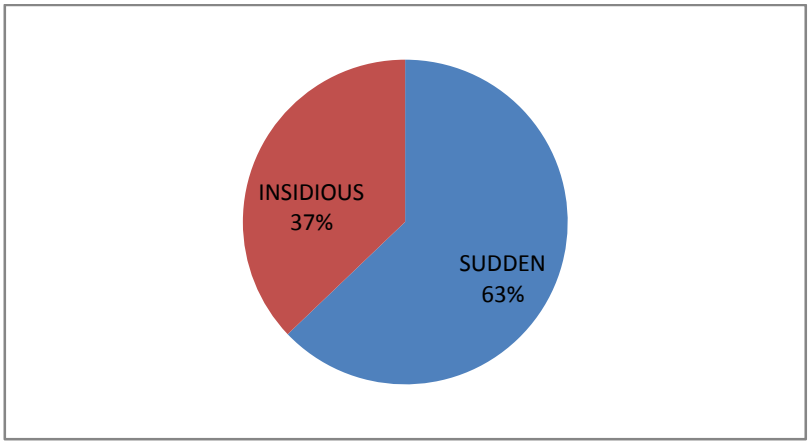
5. MODE OF ONSET

TABLE 6

MODE	FREQUENCY	%
SUDDEN	22	62.86
INSIDIOUS	13	37.14
TOTAL	**35**	**100**

In our study, 22 subjects constituting 62.86% presented with acute onset of symptoms. Whereas 13 patients (37.14%) had an insidious onset of the condition.

GRAPH-5 MODE OF ONSET



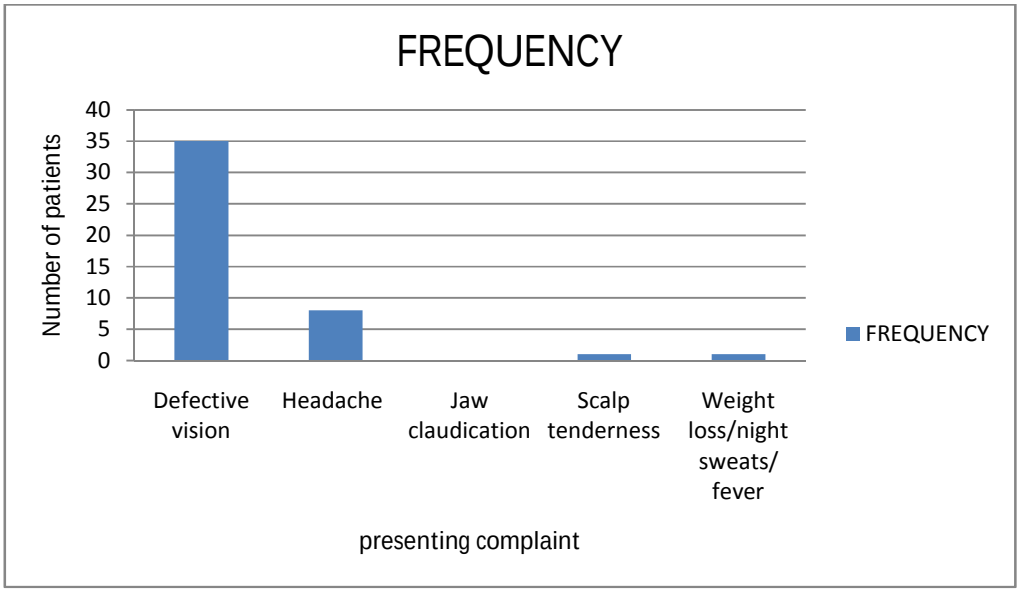
6. PRESENTING SYMPTOMS

TABLE 7

PRESENTING SYMPTOM	FREQUENCY	%
Defective vision	35	100
Headache	8	22.86
Jaw claudication	0	0
Scalp tenderness	1	2.86
Weight loss/night sweats/ fever	1	2.86
Proximal myalgia	0	0

All the subjects included in the study presented with defective vision. 8 patients (22.86%) complained of headache.

GRAPH- 6: PRESENTING SYMPTOMS



One patient (2.86%) had features of scalp tenderness along with fever, weight loss and night sweats. These features are usually associated with AAION.

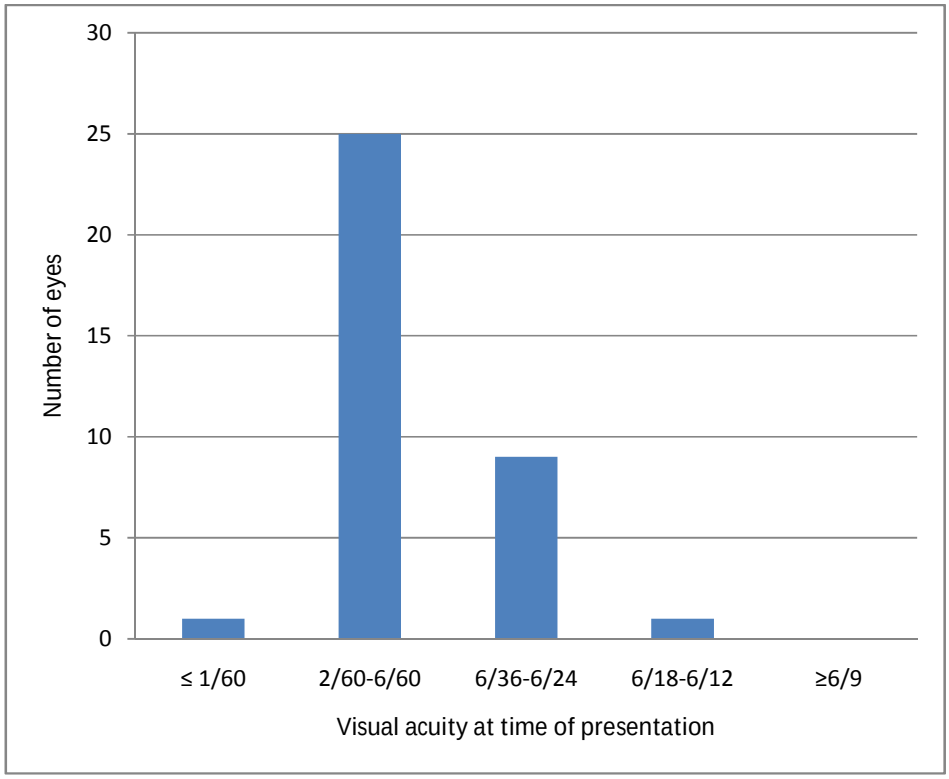
7. VISUAL ACUITY AT PRESENTATION

TABLE 8

VISUAL ACUITY	FREQUENCY	%
≤ 1/60	1	2.78
2/60-6/60	25	69.44
6/36-6/24	9	25
6/18-6/12	1	2.78
≥6/9	0	0
TOTAL NO. OF EYES	**36**	**100**

Out of the 36 eyes that were studied, 25 eyes had a visual acuity at the time of presentation in the range of 2/60-6/60. This constituted a maximum of 69.44 %. The next group consisted of eyes with visual acuity between 6/24 to 6/36 (25%). One patient each presented with visual acuity between 6/12-6/18 and less than 1/60.

GRAPH 7: VISUAL ACUITY AT THE TIME OF PRESENTATION



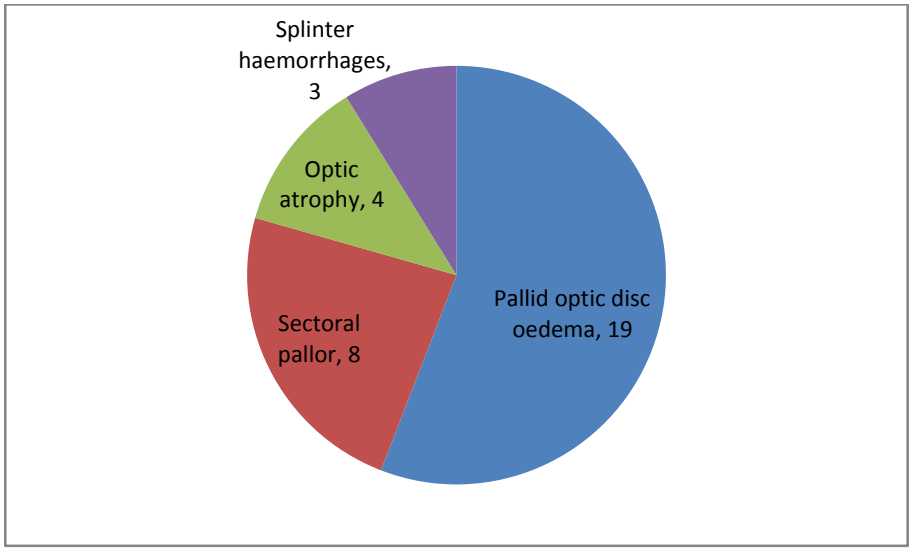
8. FUNDUS FINDING ON OPHTHALMOSCOPIC EXAMINATION AT THE TIME OF PRESENTATION

TABLE-9

Fundus finding	Frequency	%
Pallid optic disc oedema	19	54.2
Sectoral pallor	8	22.86
Optic atrophy	4	11.42
Splinter haemorrhages	3	8.57

The most common ophthalmoscopic sign at presentation was pallid disc oedema seen in 19subjects (54.2%). This was followed by sectoral disc pallor seen in 8 subjects constituting 22.86%. Splinter haemorrhages were observed in 3 subjects (8.57%) and 4 patients (11.42%) were seen to have already developed optic atrophy.

GRAPH-8: FUNDUS FINDINGS AT PRESENTATION



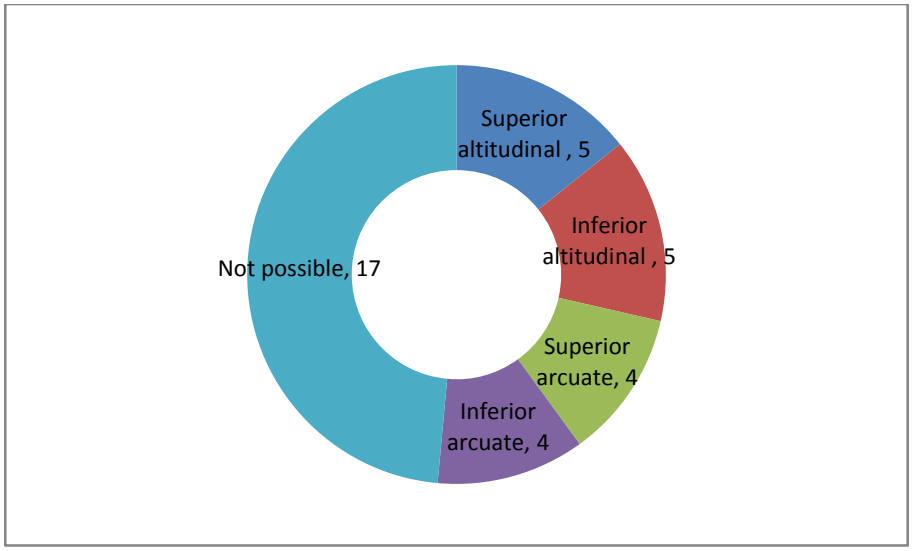
9. VISUAL FIELD DEFECTS ON AUTOMATED PERIMETRY AT THE TIME OF PRESENTATION

TABLE 10

FIELD DEFECT	FREQUENCY	%
Superior altitudinal	5	14.29
Inferior altitudinal	5	14.29
Superior arcuate	4	11.43
Inferior arcuate	4	11.43
Not possible	17	48.57

Due to poor visual acuity at the time of presentation, in 48.57% of the patients, evaluation of visual fields by automated perimetry was not possible. 5 patients each were found to have superior and inferior altitudinal field defects. 4 patients each had superior and inferior arcuate field defects.

GRAPH 9: VISUAL FIELD DEFECTS AT THE TIME OF PREENTATION



10. INVOLVEMENT OF THE FELLOW EYE

TABLE 11

PREVIOUS EPISODE IN FELLOW EYE	PATIENTS
PRESENT	1
ABSENT	34
TOTAL	35

Out of the 35 subjects, only one patient reported involvement of the other eye with similar complaints earlier. The patient was a middle aged female with history of diabetes mellitus and hypertension.

Treatment

After ocular and systemic examination, all patients underwent relevant investigations. Following this, they were started on intravenous methyl prednisolone 1 gram in two divided doses for three days followed by oral prednisolone 60 mg once daily. This was continued for one week and tapered by ten milligrams per week and stopped after a period of 6 weeks.

11. VISUAL OUTCOME FOLLOWING TREATMENT

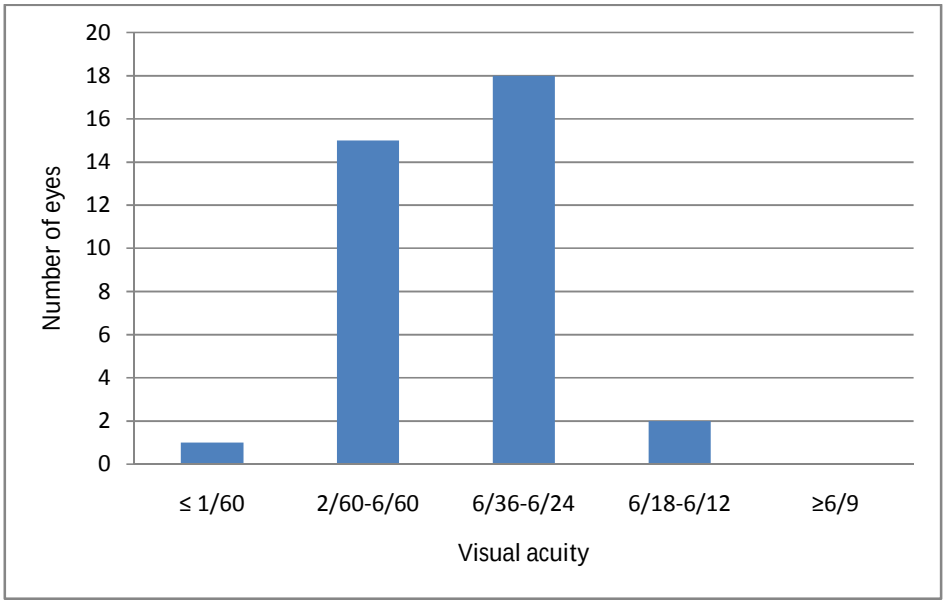
TABLE 12

VISUAL ACUITY	FREQUENCY	%
≤ 1/60	1	2.78
2/60-6/60	15	41.67
6/36-6/24	18	50
6/18-6/12	2	5.56
≥6/9	0	0
TOTAL NO. OF EYES	**36**	**100**

After starting on treatment, visual acuity measured at the end of six months showed that – 18 out of the 36 treated eyes (50%) had a visual acuity between 6/24 to 6/36.

41.67% of the patients had a final visual acuity between 2/60 to 6/60. One patient still had a visual acuity of less than 1/60 while two patients (5.56%) recovered visual acuity up to 6/12.

GRAPH-10: VISUAL OUTCOME AFTER TREATMENT



12. VISUAL OUTCOME IN DIABETIC PATIENTS

TABLE 13

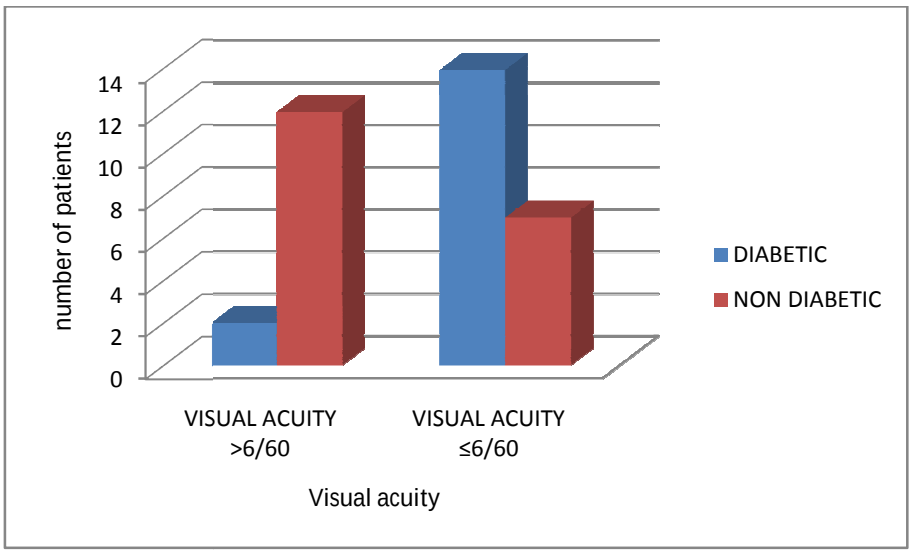
	VISUAL ACUITY >6/60	**VISUAL ACUITY ≤6/60**	**TOTAL**	**P =0.0048**
DIABETIC	2	14	**16**	
NON DIABETIC	12	7	**19**	
TOTAL	**14**	**21**	**35**	

Out of the 35 subjects included in the study, 16 patients were diabetic and 19 were non-diabetic. On comparing the visual outcome among both the groups it was observed that, only 2 out of the 16 subjects with diabetes mellitus improved with a visual acuity of more than 6/60 constituting 12.5%. A majority of 14 of the diabetics (87.5%) did not have improvement in visual acuity following treatment.

Among the 19 non-diabetic individuals, 12 of them had an improvement in visual acuity (63.16%) to more than 6/60. Only 7 of the subjects (36.84%) did not have any visual improvement.

P value is 0.0048 which is statistically significant. Thus visual outcome was poorer in diabetic than non-diabetic individuals.

GRAPH-11: COMPARISON OF VISUAL OUTCOME AMONG DIABETIC AND NON-DIABETIC PATIENTS



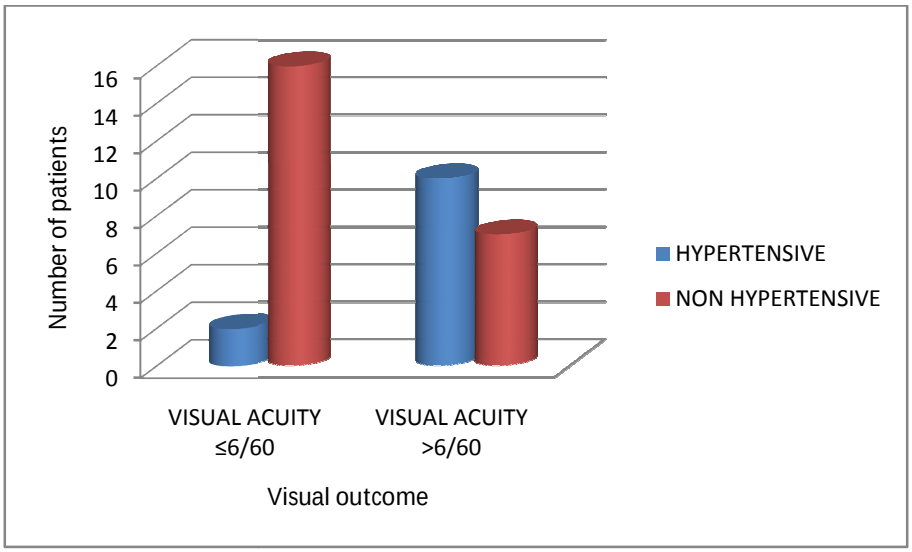
13. VISUAL OUTCOME AMONG HYPERTENSIVE PATIENTS

TABLE 14

	VISUAL ACUITY ≤6/60	**VISUAL ACUITY >6/60**	**TOTAL**	**P =0.199**
HYPERTENSIVE	2	10	**12**	
NON HYPERTENSIVE	16	7	**23**	
TOTAL	18	17	**35**	

Among the 35 patients, 12 were known hypertensive patients on treatment. 2 out of these 12 patients (16.67%) had a final visual outcome of less than 6/60. The remaining 10 (83.33%) had a final visual acuity of more than 6/60.

Among the 23 patients who did not have hypertension, 16 of them (69.57%) had a visual acuity of less than 6/60 even after treatment. While the remaining 7 patients (30.43%) had a better visual outcome.


16
14
12
10
8
6
4
2
0
Number of patients
VISUAL ACUITY ≤6/60
VISUAL ACUITY >6/60
Visual outcome
HYPERTENSIVE
NON HYPERTENSIVE

DISCUSSION

DISCUSSION

1. AGE

In this study, 35 patients with anterior ischaemic optic neuropathy were included. 57.14 % of the patients belonged to the age group of 51- 60 years. 22.86% belonged to the age group of 61-70 years.

Mean age of onset was around 56.9 years. The oldest patient was 71 years old and the youngest patient was aged 41years.

AION is typically a disease the middle aged and the elderly population with most of the studies reporting a maximum incidence in 60- 70 years age range[17]. Younger patients presenting with symptoms of this disease must be thoroughly evaluated for the presence of systemic and ocular risk factors which need to be addressed immediately to protect the fellow eye from getting involved.

2. SEX DISTRIBUTION

In our study, there was a significant gender difference with 77.14% of the patients being female while only 22.85% were males. In most of the previous studies, equal incidence rates were noted among male and female subjects[18].

3. LATERALITY

Our study showed a predominance of right eye over left eye which does not co-relate with previous studies. Only 2.86% percent of the study population gave history of involvement of the fellow eye with similar complaints.

In the ischaemic optic neuropathy decompression trial (IONDT), it was observed that 23% of subjects had a pale optic disc in the contra lateral eye which suggested a possibility of previous episode of AION.

4. ASSOCIATED RISK FACTORS

34.28% of the patients with AION were known diabetics on treatment. Systemic hypertension was also a risk factor constituting 25.71 % of the patients. 11.43% of the subjects had both diabetes and hypertension. 5.71% were anaemic. 22.86% of the subjects did not have any of the common identified risk factors.[19]

Other studies [19]reported association of diabetes mellitus in 10 to 31%, systemic hypertension among 26 to 47% of the subjects.

Thus diabetes was more predominant association among our patients.

5. MODE OF ONSET

62.86% of the patients had an acute onset of painless loss of vision which is a characteristic feature of this disease.

6. VISUAL ACUITY AT THE TIME OF PRESENTATION

All patients in the study presented with defective vision. The visual loss was most commonly sudden in onset, painless and was in the range of 2/60 to 6/60 in 69.44%. This was comparable to other studies conducted by Atkins et al which reported similar visual acuity at the time of presentation in 35 to 53% of the patients included.

7. OPHTHALMOSCOPIC FINDINGS

54.2% of the subjects had a pallid disc oedema followed by 22.86% patients having sectoral disc pallor. Splinter haemorrhages were observed in 8.57% of the subjects.

8. VISUAL FIELD DEFECTS ON AUTOMATED PERIMETRY AT THE TIME OF PRESENTATION

Due to poor visual acuity at the time of presentation, it was not possible to examine visual fields in 48.57% of the subjects.

Among the patients in whom visual field examination was possible and the fields were reliable, 27.77% showed superior and inferior altitudinal field defects respectively. 22.22 % showed superior and inferior arcuate field defects.

The classic presentation of visual field defects described in literature is an inferior altitudinal field defect. Central scotomas, quadrantic or arcuate defects may also be observed. Hayreh and Podhajsky have reported[20] inferior altitudinal field defects in 57% of their study subjects.

9. VISUAL OUTCOME FOLLOWING TREATMENT

50% of the patients included in the study reported an improvement in visual acuity between 6/24 and 6/36.

41. 67% still had a poor visual outcome of <6/60. In a study reported by Atkins et al, more than half of the subjects ended up with a vision of <6/60 at the end of the follow up period.

Among the diabetic patients included in the study, it was observed that 87.5% did not have any significant improvement in visual acuity. 63.67%

of the non diabetic subjects showed an improvement >6/60. The p value by Pearson's Chi square test was found to be 0.0048 which is statistically significant. This means that the presence of diabetes mellitus among patients with AION is associated with a poorer visual outcome than in patients without it.

Hayreh and Zimmermann reported that final visual acuity did not differ significantly among patients with and without diabetes.

Among the patients with systemic hypertension, 16.67% had a final visual outcome of less than 6/60. 83.33% had a visual acuity better than 6/60. P value was found to be more than 0.05. Hence the association was not statistically significant.

CONCLUSION

CONCLUSION

1. Anterior ischaemic optic neuropathy affects the middle aged and elderly population, with 51 to 60 years being the most affected age group in our study.
2. Females were more commonly affected than males. Female to male ratio was around 3.5:1.
3. There was a predominance of involvement of right eye over that of left eye in our study.
4. Only 2.86% of the patients showed optic disc pallor in other eye due to previous episode of AION.
5. Diabetes mellitus and systemic hypertension are associated risk factors with the development of AION. Diabetes was more predominant risk factor in our study.
6. 69.44% of the patients presented with a very poor visual acuity in the range of 2/60 to 6/60.
7. Pallid disc oedema was the most common ophthalmoscopic finding followed by sectoral pallor of optic disc and splinter haemorrhages.
8. It was not possible to do an automated perimetry to evaluate visual fields in 48.57% of the patients due to poor visual acuity at the time of presentation.

9. Among the patients in whom it was possible, superior and inferior altitudinal field defects were found in equal number of patients.
10. In 67% of the patients, visual acuity remained less than 6/60 in spite of timely treatment with corticosteroids.
11. The presence of diabetes mellitus is associated with a poor visual outcome following treatment.
12. Presence of hypertension/anaemia/ hyperlipidemia did not affect the final visual outcome.

Anterior ischaemic optic neuropathy is associated with a poor visual outcome in spite of timely intervention with corticosteroids.

It should always be considered in the differential diagnosis of painless loss of vision associated with optic disc oedema. All patients must be thoroughly evaluated for systemic risk factors and treated promptly to prevent the occurrence of ischaemic optic neuropathy in the other eye.

PART III

PROFORMA

Clinical study on Anterior ischaemic optic neuropathy

Name –	
Age –	Sex –
O.P/I.P No. –	
Date	
Address –	

Contact No. –

Unit –

Diagnosis –

Chief complaints –

1. Loss of vision – Onset -
Duration –
Progression –
Associated pain –
2. Headache –
3. Jaw claudication –
4. Scalp tenderness –
5. Similar episode in the other eye/ same eye -
6. Weight loss/ fatigue/ night sweats -

Relevant past history –

1. Diabetes mellitus - duration –
Treatment -
2. Systemic hypertension - duration –
Treatment –
3. Glaucoma –
4. Hyperlipidemia –
5. Anaemia –
6. Joint pains –
7. Drug intake –
8. Alcohol intake –
9. Smoking –
10. Fever / infections –

EXAMINATION –

General examination:

Built –

Nourishment –

Pallor –

B.P -

Cardiovascular system:

Respiratory system:

Abdominal system:

Central nervous system: higher functions

Cranial nerves

Motor system

Sensory system

Cerebellum

Examination of temporal artery region – temporal artery pulsation –

Induration –

Firmness/ nodularity of temporal artery-

OCULAR EXAMINATION -

	RE	LE
Visual acuity		
Lids		
Conjunctiva		
Cornea		
AC		
Pupil	Normal/RAPD Direct Indirect	Normal/RAPD Direct Indirect
Lens		
IOP		
Colour vision		

Fundus examination –

Direct ophthalmoscopy Media

Optic disc

CD ratio

Indirect ophthalmoscopy

Fields –

	RE	LE
Manual		
AP		
Inference		

FFA –

PROVISIONAL DIAGNOSIS –

BLOOD INVESTIGATIONS –

1. Complete haemogram

TC

DC N L M E B

ESR

Hb

2. RBS -

3. Total cholesterol –

4. C- reactive protein

Cardiologist opinion –

MANAGEMENT –

Date	Treatment	
	IVMP	
	T. Predni	
	T. Aspirin	

Follow up -

Date	Visual acuity		Field of vision		Colour vision		Fundus	Pupil
	RE	LE	RE	LE	RE	LE		
Admission								
2 weeks								
2 months								
6 months								

KEY TO MASTER CHART

M - MALE

F - FEMALE

RE - RIGHT EYE

LE - LEFT EYE

NAD - NO ABNORMALITY DETECTED

DM - DIABETIC MELLITUS

HTN - HYPERTENSION

VN - VISION

ESR - ERYTHROCYTE SEDIMENTATION RATE

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